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**Issue Date: 01 June 2005**

In the Matter of

James C. Honaker,
Claimant

v.

Sea B Mining Company,
Employer

and

Director, Office of Workers' Compensation Programs,
Party-In-Interest

Case No. 2004BLA06171

## DECISION AND ORDER AWARDING BENEFITS

This proceeding arises from a claim for Benefits under the Black Lung Benefits Act of 1977, 30 U.S.C. §901 *et seq*. (hereinafter "the Act"). In accordance with the Act and the regulations issued thereunder, the case was referred by the Director, Office of Workers' Compensation Programs for a formal hearing.

Benefits under the Act are awardable to miners who are totally disabled within the meaning of the Act due to pneumoconiosis, or to the survivors of miners who were totally disabled at the time of their deaths (for claims filed prior to January 1, 1982), or to the survivors of miners whose deaths were caused by pneumoconiosis. Pneumoconiosis is a dust disease of the lungs arising from coal mine employment and is commonly known as "black lung."

A formal hearing was held before me on February 17, 2005, in Abingdon, Virginia, at which time all parties were afforded full opportunity in accordance with the Rules of Practice and Procedure (29 C.F.R. Part 18) to present evidence and argument as provided in the Act and the regulations issued thereunder, set forth in Title 20, Code of Federal Regulations, Parts 410, 718, 725, and 727. At that hearing, I admitted into the record Director's Exhibits 1-37, Claimant's Exhibit 1-2, and Employer's Exhibits 1-4. The Claimant submitted a brief on May 27, 2005; the Employer submitted a brief on May 27, 2005; the Director did not submit a brief.

I have based my analysis on the entire record, including the transcript, exhibits, and representations of the parties, and given consideration to the applicable statutory provisions, regulations, and case law, and made the following findings of fact and conclusions of law.

## Jurisdiction and Procedural History[1]

The Claimant filed his claim for benefits on November 18, 2002 (DX 2). On January 5, 2004, the District Director, Office of Workers' Compensation Programs (hereinafter "Director") issued a Proposed Decision and Order denying benefits (DX 29). The Claimant timely requested a hearing, and the matter was then referred to the Office of Administrative Law Judges on April 23, 2004 (DX 35). The claim was assigned to me, and I conducted a formal hearing on February 17, 2005.

## Findings of Fact and Conclusions of Law

### Issues

The only issue contested by the Employer and Director is whether the Claimant is totally disabled due to pneumoconiosis (DX 35; Tr. 10-12).

### Stipulations

The parties have agreed that: (1) the Claimant is a miner under the Act; (2) the Claimant engaged in post-1969 coal mine employment; (3) the Claimant has 26.59 years of coal mine employment experience; (4) the Claimant has pneumoconiosis; (5) the Claimant has one dependent for purposes of augmentation of benefits; and (6) the Employer is properly named as the Responsible Operator (DX 35; Tr. 10-12).

### Background

The Claimant was born on October 17, 1953; he has a twelfth grade education. The Claimant is married to his wife Deborah Sue, who is his sole dependent (DX 9). The Claimant was previously married to Judy M. Puckett; they divorced on July 16, 1984 (DX 2, 10).

## APPLICABLE STANDARD

Because this claim was filed after the enactment of the Part 718 regulations, the Claimant's entitlement to benefits will be evaluated under Part 718 standards. In order to establish entitlement to benefits under Part 718, the Claimant must prove that he has pneumoconiosis, that it arose out of his coal mine employment, and that the pneumoconiosis has caused him to be totally disabled.

*Medical Evidence*

The following medical evidence is in the record.[2]

[1] Citations to the record of this proceeding will be abbreviated as follows: "Tr." refers to the Hearing Transcript of the February 17, 2005 hearing; "ALJX" refers to the Administrative Law Judge's Exhibits; "DX" refers to the Director's Exhibits; "CX" refers to Claimant's Exhibits; and "EX" refers to Employer's Exhibits.

## X-ray Evidence[3]

| *Exhibit No.* | *Date of X-ray* | *Reading Date* | *Physician/ Qualifications* | *Impression* |
|---|---|---|---|---|
| DX 28* | 8-27-74 | 10-28-74 | Wiot/B, BCR | Negative |
| DX 28* | 1-3-79 | 4-2-79 | Cole/B, BCR | Negative |
| DX 28* | 9-5-90 | 9-5-90 | Patel | Negative |
| DX 28* | 9-5-90 | 10-30-90 | Gordonson/BCR | Negative |
| DX 28* | 12-11-91 | 12-11-91 | Patel | Negative |
| DX 28* | 12-11-91 | 2-15-92 | Proto/BCR | Negative |
| DX 28* | 4-24-98 | 6-16-98 | Wheeler/B, BCR | Negative for pneumoconiosis |
| DX 13 | 12-05-02 | 12-24-02 | Navani/B, BCR | Read for quality purposes only |
| DX 13 | 12-05-02 | 12-20-02 | Forehand/B | 1/2, q, q |
| DX 28 | 7-21-03 | 7-22-03 | Hippensteel/B | 2/1, q, q |
| EX 2 | 8-11-04 | 8-12-04 | Castle/B | 2/1, q, q |

## Pulmonary Function Studies

| *Exhibit No.* | *Date* | *Age/Ht* | *FEV1* | *FVC* | *MVV* | *Effort* |
|---|---|---|---|---|---|---|
| DX 13 | 12-5-02 | 49/70” | 3.24 | 3.99 | 93 | Good |

2 In its evidence summary form, the Employer identified seven x-ray readings, all negative, under the category for hospitalization records and treatment notes. Each of these interpretations is on an ILO form; I have identified them with an asterisk. While the interpretations may in fact be of x-rays performed while the Claimant was hospitalized, or in connection with treatment, there are no such hospitalization records or treatment notes in the record. These x-ray interpretations are not part of any hospitalization records or treatment notes, nor are there any treatment notes or hospitalization records that they appear to rebut or address. Thus, they are not admissible under 20 C.F.R. § 725.414(a)(4)(2001), and I have not considered them. As the Employer has conceded the issue of pneumoconiosis, their relevance is questionable.

3 B-B reader; and BCR - Board Certified Radiologist. These designations indicate qualifications a person may possess to interpret x-ray film. A “B Reader” has demonstrated proficiency in assessing and classifying chest x-ray evidence for pneumoconiosis by successful completion of an examination. A “Board Certified Radiologist” has been certified, after four years of study and an examination, as proficient in interpreting x-ray films of all kinds including images of the lungs.

| *Exhibit No.* | *Date* | *Age/Ht* | *FEV1* | *FVC* | *MVV* | *Effort* |
|---|---|---|---|---|---|---|
| DX 28 | 7-21-03 | 49/70" | 2.75<br>2.95 | 3.51<br>3.65 | 77 | |
| EX 2 | 8-11-04 | 50/70" | 2.62<br>2.53 | 3.49<br>3.25 | 95 | |

**Arterial Blood Gas Studies**

| *Exhibit No.* | *Date* | *Physician* | *pCO2* | *pO2* | *At rest/exercise* |
|---|---|---|---|---|---|
| DX 13[4] | 12-5-02 | Forehand | 36<br>38 | 85<br>61 | At rest<br>After Exercise |
| DX 28 | 7-21-03 | Hippensteel | 38.2<br>34.7 | 83.7<br>60.9 | At rest<br>After exercise |
| EX 2 | 8-11-04 | Castle | 39.2<br>36.4 | 84.1<br>64.1 | At rest<br>After exercise |

**Medical Opinion Evidence**

*Dr. J. Randolph Forehand*

Dr. Forehand examined the Claimant at the request of the Department of Labor on December 5, 2002 (DX 13). He noted the Claimant's history of coal mine employment, as well as his family and medical histories. Dr. Forehand reported that the Claimant smoked a pack of cigarettes a day from 1972 to 2000. On examination of the Claimant, Dr. Forehand noted diminished breath sounds, and crackles at the bases. The Claimant's x-ray reflected coal workers' pneumoconiosis. Although his pulmonary function studies showed a normal ventilatory pattern, his arterial blood gas studies showed hypoxemia with exercise, and no metabolic disturbance. There were no acute changes in his EKG.

Based on the results of the x-ray, the physical examination, the results of the arterial blood gas studies, and the Claimant's work history, Dr. Forehand concluded that he has coal workers' pneumoconiosis, due to his exposure to coal dust. According to Dr. Forehand, the Claimant has a significant respiratory impairment of a gas exchange nature, and he has insufficient residual oxygen transfer capacity to return to his last coal mining work. Dr. Forehand stated that the Claimant is unable to work, and is totally and permanently disabled. According to Dr. Forehand, coal workers' pneumoconiosis is the sole factor contributing to the

[4] Dr. Michos reviewed these results and determined that they were technically acceptable (DX 13).

Claimant's respiratory impairment. He stated that there was no evidence of emphysema from cigarette smoking or heart failure.

Dr. Forehand also testified at the hearing. His testimony is attached to this decision, and incorporated by reference. Dr. Forehand discussed his background and training, stating that he completed training in the field of pediatrics, and practiced pediatrics for six years, until 1984, in the coal fields of southwest Virginia. During this time, working with families of coal miners, he developed an interest in pulmonary disease, and decided to leave pediatrics (Tr. 15).

Dr. Forehand began training in allergy and pulmonary medicine at the National Jewish Hospital in Denver, Colorado. The training consisted of clinical training, and research in allergy and pulmonary medicine. He then joined the faculty at the University of Pennsylvania, attending and caring for patients with allergic and pulmonary diseases (Tr. 16).

Dr. Forehand described the process for board certification in a medical specialty. He stated that the training program director requests that a physician be allowed to sit for a specialty or sub-specialty board. However, Dr. Forehand's background and certification is in pediatrics, and he would not be allowed to sit for the adult pulmonary treatment boards.

In 1990, Dr. Forehand returned to southwest Virginia. Because of his interest in pulmonary medicine and mechanics, the Department of Labor asked him to be a medical examiner for the black lung program. He has performed in this role for fifteen years, and has examined more than 4,000 miners (Tr. 17-18). He has also reviewed medical files at the request of the Department of Labor.

Dr. Forehand has not practiced pediatric medicine since 1984. Since that time, he has practiced exclusively in the field of allergy and pulmonary medicine (Tr. 18-19). He is currently a staff physician, and the medical director of the cardiopulmonary diagnostic laboratory at the clinic. According to Dr. Forehand, he has performed all of the requirements necessary to sit for the pulmonary certification board, and has taken the 60 hours of continuing medical education required by the board every two years (Tr. 19). He also keeps current with publications, the primary ones being published by the American Thoracic Society, and the American College of Chest Physicians. He has also submitted abstracts many times on his clinical research to the American Thoracic Society, and presented his abstracts at the annual meeting of the American Thoracic Society, the scientific arm of the American Lung Association (Tr. 20).

Dr. Forehand testified that about 80 percent of the men he sees in his practice are or were coal miners.

Dr. Forehand discussed Dr. Hippensteel's first report; he did not agree that there was a relationship between the Claimant's respiratory impairment and the possibility of ischemic heart disease. Dr. Forehand suggested that the Claimant see Dr. Piriz, a cardiologist. Dr. Forehand agrees with the results of Dr. Piriz's testing, which showed that the Claimant did not have ischemic heart disease, or any type of work related impairment that might arise from heart disease (Tr. 23)..

Dr. Forehand described hypoxemia as a lack of sufficient oxygen. Causes include the inability of the heart to pump blood to the lungs to be oxygenated, which could be caused by heart disease. If a person stops breathing, for example due to severe head trauma, and oxygen is not being brought into the body, that is another form of hypoxemia.

In another form of hypoxemia, a diffusion block, oxygen is brought into the lung, into the air sacs or alveoli, but the space between the alveoli and the pulmonary blood vessels has become widened, so that the oxygen cannot diffuse across the gap. This form of hypoxemia is called a diffusion block.

Another cause of hypoxemia is when blood circulates through one part of the lung, but the person is ventilating a separate part of the lung, so that the circulating blood never reaches the alveoli. This form of hypoxemia is called a ventilation perfusion mismatch (Tr. 24).

Based on the results of the Claimant's testing, as well as his personal experience testing over 4,000 miners for the Department of Labor, and his reading of medical literature, Dr. Forehand concluded that the Claimant has a ventilation perfusion mismatch, which was the cause of his hypoxemia (Tr. 25).

Dr. Forehand explained that it is possible to test for a diffusion block, with a test called DLCO. This tests for the integrity of the alveoli or capillary membrane or gap space. However, because hypoxemia due to coal dust exposure is not due to a diffusion block, he would not expect to see an abnormal DLCO. Hypoxemia due to coal workers' pneumoconiosis is caused by a ventilation perfusion mismatch: when the coal dust is inspired in the lung, macrophages or phagocytes pick the particles up, and try and return them into the circulation through the lymphatics, which then become clogged. Scarring then occurs around the blood vessels where the lymphatics run parallel. The number of functional blood vessels in the lung is reduced, and although the lung is being ventilated, it is not perfusing as well (Tr. 26). This is a ventilation perfusion mismatch. Because the scarring is not occurring at the alveolar capillary bed, the DLCO is not abnormal (Tr. 27).

Dr. Forehand discussed a study of lung impairment in non-smoking coal miners, which showed that the DLCO is normal in the presence of coal dust induced hypoxemia (Tr. 27; CX 2). Noting that the Claimant is a smoker, Dr. Forehand stated that cigarette smoking causes emphysema, and a breakdown of the alveolar capillary interface, resulting in hypoxemia, and an abnormal DLCO. However, the Claimant does not have obstructive lung disease or emphysema (Tr. 28).

According to Dr. Forehand, the study used non-smoking miners, to remove the confounding factor of cigarette smoking. The miners were hypoxic, but they had normal DLCO's. The study showed that the reason for their hypoxia was an uneven ventilation of perfusion, as he described above (Tr. 28). In other words, an abnormal DLCO would be present with a perfusion block, but not in mixed ventilation perfusion (Tr. 29).

This study appeared in the European Respiratory Journal in 1988. However, the miners in the study were West Virginia coal miners. Dr. Forehand was not aware of any study that

challenged or altered the results of this study (Tr. 29-30). According to Dr. Forehand, the European Respiratory Journal is the official publication of the European Respiratory Society. As it is often referenced in textbooks of occupational medicine, to explain the mechanism of hypoxemia in coal miners, he would expect pulmonologists or anyone who works in the area to be familiar with this study (Tr. 30). He did not see any reference to the article in Dr. Hippensteel's deposition.

Dr. Forehand was asked about Dr. Hippensteel's statement that according to the medical literature, more than 50 percent of persons with simple coal workers' pneumoconiosis do not have any ventilatory impairment. He was not aware of any such literature or studies, or any textbooks that had made such statements (Tr. 31).

On cross-examination, Dr. Forehand was asked about the size of the study, 20 miners. He stated that it was hard, in a study of this sophistication, to get 20 persons to travel from West Virginia to New York, and undergo an invasive study (Tr. 32). He did not think that the number of persons in the study mattered.

When it was pointed out to Dr. Forehand that the persons in the study were not exercised, Dr. Forehand stated that the point of the study had nothing to do with exercise induced impairment. Rather, it involved a determination of what coal dust does to miners. Even though a miner may not be complaining or overtly short of breath, that does not mean that there is not a pathologic process going on – that is what the study was designed to show (Tr. 35-37).

Dr. Forehand testified that he is not using the study to show that a ventilation perfusion mismatch causes a drop in p02 with exercise. Rather, he was concerned about Dr. Hippensteel's testimony that, because the Claimant has a normal DLCO, he cannot have hypoxemia; he must have heart disease (Tr. 37). In his view, this statement is wrong (Tr. 44).

Dr. Forehand stated that the study to which he referred was of grand scale; he was not surprised that there were no more recent studies. He testified that he did not think that medical science felt the need to confirm or refute the results. It was a good study, published in a reputable journal, by authors who are knowledgeable of coal workers' pneumoconiosis and pulmonary mechanics; there probably was no reason to do additional studies (Tr. 39-40).

Dr. Forehand agreed that the Claimant had normal ventilatory function. He stated that in his personal experience, coal miners with exercise induced hypoxemia from coal workers' pneumoconiosis often have normal ventilatory function. But there is no simple relationship between the degree of abnormality on x-ray and the degree of abnormality of pulmonary function (Tr. 40).

Dr. Forehand acknowledged that hypoxemia with a ventilation perfusion mismatch, and a normal DLCO, could be reversible. But this was not the situation with the Claimant. He indicated that he sees miners all the time, who have a sedentary lifestyle, and have a low p02 at rest. They are perfusing the top and ventilating the bottom, or vice versa. But once they are exercised, and increase their cardiac output, their p02 normalizes. This is an example of reversibility of perfusion ventilation defects. But with the Claimant, it was just the opposite. His

p02 at rest was normal. Since the Claimant indicated that he gets short of breath with exercise, but not at rest, he was exercised. His p02 was abnormal with exercise (Tr. 44-45). Indeed, Dr. Forehand stated that the Department of Labor instructs physicians to perform a blood gas study at rest, and if it is normal, to perform an exercise study, because there are a certain number of miners who will only be impaired with exercise (Tr. 45).

*Dr. Kirk E. Hippensteel*

Dr. Hippensteel examined the Claimant at the request of the Employer on July 21, 2003 (DX 28). He reported the Claimant's history of coal mine employment, as well as his family and medical histories. Dr. Hippensteel reported that the Claimant started smoking at age 18, averaging about a pack of cigarettes a day, and currently smoked about one and a half packs a day.

On examination of the Claimant, Dr. Hippensteel noted good breath sounds bilaterally, with no rales or wheezes with forced expiration. The Claimant's heart rhythm was regular, with no gallop or murmur. The Claimant's chest x-ray was classified as 2/1, q, q, in all lung zones. The results of the Claimant's spirometry testing showed no obstruction. According to Dr. Hippensteel, the results of the MVV testing were invalid, due to suboptimal effort. Lung volumes were normal, indicating no restriction; diffusion was normal.

The Claimant's resting electrocardiogram was normal. His resting arterial blood gas test results were normal; his carboxyhemoglobin level was elevated, consistent with his current smoking. Dr. Hippensteel had the Claimant exercise on a treadmill, and during this exercise he developed some ST depression. He had no chest pain, but he had a drop in his p02, indicative of mild hypoxemia. According to Dr. Hippensteel, this was further aggravated by apparent smoking between the resting and exercise blood gas testing, as his carboxyhemoglobin level went up.

Dr. Hippensteel stated that with normal diffusion, and electrocardiographic changes associated with exercise, it was possible to state that the Claimant's hypoxemia was cardiac rather than pulmonary related.

Dr. Hippensteel concluded that there was radiographic evidence for a "possible" diagnosis of coal workers' pneumoconiosis, but there was no ventilatory or gas exchange impairment that could be ascribed to pneumoconiosis. According to Dr. Hippensteel, the Claimant has evidence of cardiac ischemia with exercise, not associated with chest pain.

Dr. Hippensteel also reviewed Dr. Forehand's report, as well as the test results he obtained. He stated that Dr. Forehand was not an expert in heart diseases in adult patients, since he is a pediatrician allergist. According to Dr. Hippensteel, Dr. Forehand failed to comment on electrocardiographic findings with exercise, which was a significant omission.

Dr. Hippensteel felt that with the more complete data he obtained on his examination of the Claimant, he found that the Claimant had evidence of a gas exchange impairment, but no diffusion impairment, findings which are against any lung origin for the gas exchange

impairment with exercise. Dr. Hippensteel also found evidence of cardiac dysfunction with exercise, which made cardiac function the cause for the Claimant's gas exchange impairment. According to Dr. Hippensteel,

> These findings of a more complete nature on my examination show how one needs to have a proper set of data before one can make correct conclusions about cause of impairment. Gas exchange impairment is not just associated with lung diseases, but is also associated with cardiac dysfunction, as was shown to be the case on my examination of this man.

Dr. Hippensteel also noted that the x-ray changes were very recent, which made it possible that they were not related to coal workers' pneumoconiosis, but to some other disease, including granulomatous disease.[5] But even if it were stipulated that the Claimant's x-rays represented a manifestation of simple coal workers' pneumoconiosis, Dr. Hippensteel felt that the Claimant did not have any ventilatory or gas exchange impairment as a result. Rather, Dr. Hippensteel felt that the Claimant had developed exercise induced gas exchange impairment related to heart dysfunction. This is confirmed by his normal diffusion. Dr. Hippensteel stated:

> Dr. Forehand did not even include cardiac disease as a potential source of his gas exchange impairment and used far less data than I have used on which to base his conclusions, which, in my opinion, are improper, based upon the reasons I have stated above.

According to Dr. Hippensteel, the Claimant is disabled as a whole man, but not from a pulmonary standpoint.

Dr. Hippensteel testified by deposition on February 15, 2005 (EX 1). He had had the opportunity to review test results obtained by Dr. Castle, which showed a mild reduction in FEV-1 and FVC on spirometry, and normal lung volumes and diffusion. Dr. Castle also noted similar S-T segment depression during exercise.

Dr. Hippensteel testified that, based on the pulmonary function test results, the Claimant had no evidence of any ventilatory or diffusion impairment.

Dr. Hippensteel reviewed records from the Clinch Valley Medical Center dated August 30 and 31, 2004. These included a progress record from Dr. Piriz, an EKG, and the results of an exercise test. Dr. Hippensteel noted that Dr. Forehand had referred the Claimant for a cardiac evaluation, based on his complaints of chest discomfort at night, and his fatiguability and shortness of breath on exertion. At this evaluation, the Claimant performed a treadmill stress test, which produced a normal response to exercise. Dr. Piriz concluded that the findings did not suggest any underlying ischemia or previous myocardial infarction.

Dr. Hippensteel felt that the results obtained by Dr. Piriz make it unlikely that the Claimant had ischemic changes in his heart function at the time of exercise. However, according to Dr. Hippensteel, the results do not rule out some alteration in cardiac output that would have

[5] In this regard, Dr. Hippensteel reviewed five x-ray interpretations spanning the years from 1974 to 1998.

an impact on his gas exchange. The Claimant has normal diffusion capacity, which shows that the diffusion of oxygen across his lung tissue is in the normal range, and not indicative of impairment from lung disease.

Dr. Hippensteel discussed the different causes for a drop in p02 with exercise. He described one as impairment in diffusion across the alveolar membrane, which allows oxygen to pass less freely than normal into the bloodstream with exercise. Or there could be effects from the heart not pushing blood through the lungs as fast to pick up the oxygen from the air sacs. There could be abnormalities in the blood itself that is not exchanged, such as anemia, which would affect how much oxygen could be picked up as the blood passes through the lungs. There could also be abnormalities in the hemoglobin, causing the blood not to pick up oxygen as well during a stressed state such as exercise.

According to Dr. Hippensteel, coal workers' pneumoconiosis can cause reductions in arterial blood gas study results, by affecting diffusion. He stated that if there is associated airways disease, it can affect ventilation perfusion matching as well. He described this as a process where the gas exchange is affected at rest, and improves with exercise. According to Dr. Hippensteel, "Diffusion impairment is what is typically associated with lung diseases that affect impairment with gas exchange with exercise, as happened in this man, and this man does not have diffusion impairment."

Dr. Hippensteel acknowledged that coal dust could aggravate obstructive lung disease and cause abnormalities in blood gases. But the Claimant does not have obstructive lung disease. According to Dr. Hippensteel, there is no objective evidence that the Claimant's pneumoconiosis on x-ray plays any role in his drop in p02 with exercise. He noted that all of the examinations reflected normal ventilatory function, and normal diffusion. Thus, his gas exchange abnormalities with exercise are not due to a diffusion impairment.

Dr. Hippensteel testified that "it appears that this is the case," that the Claimant's problems with decreased p02 with exercise are due to causes that none of the tests would show. He again stated that the Claimant has no respiratory impairment, although he does have an impairment in gas exchange, that qualifies him as disabled under the Black Lung regulations. But this impairment is not related to his exposure to coal dust.

Dr. Hippensteel stated that it would be possible to determine the heart problems that are causing the Claimant's drop in p02 with exercise with an echocardiogram with exercise, or a blood function test to determine the type of hemoglobin he has. But he also stated that even if those tests produced a normal response, he would feel that there was no answer as to why the Claimant's gas exchange was abnormal.

According to Dr. Hippensteel, the presence of an "alveolar capillary block," or a relative blockage of a passage of the oxygen across a membrane thickened by interstitial inflammation in pneumoconiosis, can be ruled out by the fact that the Claimant has a normal diffusion capacity.

Dr. Hippensteel testified that persons with a reading of 2/1 very frequently do not have any interference in their lung function, and that in looking at impairments overall, less than 50

percent have such impairment. He claimed that he based this on the "medical literature," in other words, textbooks on occupational pneumoconiosis.

*Dr. James R. Castle*

Dr. Castle examined the Claimant at the Employer's request, and provided a report dated August 26, 2004 (EX 2). He noted the Claimant's history of coal mine employment, as well as his medical and smoking histories. On examination of the Claimant, Dr. Castle reported a normal chest AP diameter, with no intercostals retractions or use of accessory muscles with quiet breathing. The Claimant had normal and equal breath sounds throughout, with no rales, rhonchi, wheezes, rubs, crackles, or crepitations.

Dr. Castle reviewed the Claimant's chest x-ray, finding profusion of q/q type opacities in all lung zones, 2/1, consistent with coal workers' pneumoconiosis. The Claimant underwent pulmonary function studies, which showed normal total lung capacity and diffusing capacity. The Claimant also underwent a resting arterial blood gas study. The Claimant's carboxyhemoglobin level was elevated. Exercise studies were attempted, but after three minutes and 41 seconds, the test was stopped because of ST depression; the Claimant did not have any pain. Blood gases were drawn while the Claimant was exercising. Dr. Castle recommended that the Claimant see his family physician for possible evaluation of heart problems.

Dr. Castle concluded that the radiographic evidence was consistent with coal workers' pneumoconiosis. The Claimant's test results showed a mild reduction in forced vital capacity and FEV1, with normal lung volumes and diffusing capacity, and mild exercise induced hypoxemia. There were exercise induced St-T wave changes of possible cardiac ischemia.

Dr. Castle also reviewed other medical records, including the reports from Dr. Forehand and Dr. Hippensteel. He noted that another risk factor for the development of pulmonary disease, in addition to the Claimant's extensive history of coal mining employment, was his 32 pack year history of smoking, which was sufficient to have caused him to develop chronic obstructive pulmonary disease, lung cancer, or atherosclerotic cardiovascular disease if he were a susceptible host. An additional risk factor for the development of shortness of breath was cardiac disease. Dr. Castle stated that the Claimant did not indicate that he had any heart disease or chest pain, but on two occasions he developed very minor or early manifestations of possible coronary artery disease. Dr. Castle noted that the Claimant did not have consistent findings of rales, crackles, or crepitation.

According to Dr. Castle, the valid physiologic studies did not show any significant functional abnormalities, in the form of airway obstruction, restriction, or diffusion abnormality. His arterial blood gases at rest were normal, although he had a significant reduction in p02 with exercise. He pointed out that Dr. Hippensteel felt that this change was due to probable ischemic heart disease. Dr. Castle felt that the minor ST-T wave changes during his exercise study of the Claimant could possibly have been caused by cardiac changes. However, based on the information he had, he could not state this with reasonable medical certainty. In fact, it was not possible for him to state with reasonable medical certainty the exact etiology of the Claimant's

exercise induced hypoxemia. He stated that if pneumoconiosis were the cause, he would expect to find other abnormalities on pulmonary testing, which were not present.

Dr. Castle stated that the Claimant does not have a disabling mechanical functional ventilatory impairment. However, he has exercise induced hypoxemia, which may be of cardiac origin, although Dr. Castle is not able to state this with reasonable medical certainty, in the absence of documented cardiac disease.

*Dr. Jose M. Piriz*

Dr. Piriz administered a treadmill stress test on August 31, 2004 (CX 1). According to Dr. Piriz, he had a normal blood pressure and heart rate response to exercise, and exhibited absolutely no symptoms throughout the test. The test was terminated after achievement of greater than 85% of maximum predicted heart rate, and because of the Claimant's complaints of fatigue.

Dr. Piriz's review of the baseline EKG showed an overall normal sinus rhythm with J-point elevation; there were no acute ischemic changes seen. Dr. Piriz's review of the EKG did not show any specific or significant changes from baseline, or malignant dysrhythmias.

Dr. Piriz also performed technetium sestamibi imaging, which showed an overall chamber size that did not change at rest. The stress perfusion imaging did not reveal any specific or significant areas of photon deficiencies, and resting images were virtually unchanged. The gated SPECT imaging showed an overall normal right ventricular cavity size, with preserved function. The left ventricle was also normal in size and systolic function. Dr. Piriz noted no specific or significant segmental wall motion abnormalities. The ejection fraction was 69%.

Dr. Piriz's impression was: (1) negative/normal EKG portion of treadmill stress testing; (2) negative/normal stress myocardial perfusion imaging; and (3) negative/normal gated SPECT imaging. According to Dr. Piriz, these findings are consistent with negative/normal stress myocardial perfusion imaging, and are not suggestive of any underlying ischemia or previous myocardial infarction.

In his office notes from the previous day, Dr. Piriz noted that the results of the Claimant's August 11, 2004 EKG tests (performed by Dr. Castle) were actually quite benign, with no evidence of any ischemic changes.

## DISCUSSION

As noted above, the only contested issue in this matter is whether the Claimant is totally disabled due to pneumoconiosis. The regulations as amended provide that a claimant can establish total disability by showing pneumoconiosis prevented the miner "[f]rom performing his or her usual coal mine work," and "[f]rom engaging in gainful employment in the immediate area of his or her residence requiring the skills or abilities comparable to those of any employment in a mine or mines in which he or she previously engaged with some regularity over a substantial period of time." 20 C.F.R. §718.204(b)(1). Total disability may be established by

pulmonary function tests, arterial blood gas tests, evidence of cor pulmonale with right-sided congestive heart failure, or physicians' reasoned medical opinions, based on medically acceptable clinical and laboratory diagnostic techniques, to the effect that a miner's respiratory or pulmonary condition prevents or prevented the miner from engaging in the miner's previous coal mine employment. 20 C.F.R. §718.204(b)(2).

The regulations at § 718.204(b)(2)(i) provide that, in the absence of contrary probative evidence, arterial blood gas tests that meet the values listed in Appendix C shall establish a miner's total disability. Here, while the Claimant's arterial blood gas test results did not meet those values at rest, the results obtained by Dr. Forehand during exercise studies did meet those values. Thus, in the absence of contrary probative evidence, these values establish that the Claimant is totally disabled.

Dr. Forehand has offered a cogent and articulate explanation of the significance of the Claimant's arterial blood gas test results with exercise. He stated that these results indicate a significant gas exchange impairment, which means that the Claimant has insufficient residual oxygen transfer capacity to perform his previous coal mining work. The Claimant does not have emphysema from his cigarette smoking, or heart failure. According to Dr. Forehand, the sole factor in his gas exchange impairment is his exposure to coal mine dust.

Dr. Forehand described hypoxemia as a lack of sufficient oxygen, and listed four different causes. He concluded that the Claimant suffers from a form of hypoxemia called ventilation perfusion mismatch, where blood circulates through one part of the lung, but a separate part of the lung is being ventilated, so that the circulating blood does not reach the alveoli. He addressed the results of the DLCO test relied on by Dr. Hippensteel, explaining that the DLCO tests for the integrity of the alveoli or capillary membrane or gap space, or in other words, for a diffusion block. But hypoxemia due to coal dust exposure is not caused by a diffusion block. It is due to scarring around the blood vessels, after coal dust is inspired in the lung, and the lymphatics become clogged when macrophages or phagocytes pick the particles up and try to return them into the circulation. This reduces the number of functional blood vessels, and although the lung is being ventilated, it is not perfusing as well. The DLCO is not abnormal, because the scarring is not at the alveolar capillary bed.

Dr. Forehand cited to a study of lung impairment in non-smoking coal miners, which demonstrated that the DLCO is normal in the presence of coal dust induced hypoxemia. According to Dr. Forehand, cigarette smoking causes emphysema, and a breakdown of the alveolar capillary interface, which results in hypoxemia, and an abnormal DLCO. But the Claimant does not have obstructive lung disease or emphysema. This study was performed in 1987, with West Virginia coal miners, and was published in 1988 in the European Respiratory Journal, the official publication of the European Respiratory Society. According to Dr. Forehand, it is often referenced in textbooks of occupational medicine, to explain the mechanism of hypoxemia in coal miners, and he would expect anyone who works in pulmonary medicine to be familiar with it.

Dr. Hippensteel has dismissed Dr. Forehand's conclusions, because he is a pediatrician, who has no experience treating adult cardiac patients. However, as Dr. Forehand testified,

although his training and practice were initially in the field of pediatrics, he has practiced exclusively in the field of allergy and pulmonary medicine since 1984. Since 1990, he has practiced in southwest Virginia, where the vast majority of his patients are current or former coal miners. In addition to his treatment of coal miners, Dr. Forehand has examined more than 4,000 miners in his role as a medical examiner for the Department of Labor for the last fifteen years.

It is accurate to state that Dr. Forehand is not board certified in pulmonary medicine. As Dr. Forehand explained, he is not allowed to sit for the adult pulmonary treatment boards, because his initial training and his certification were in pediatrics. However, other than the board certification, Dr. Forehand has performed all of the requirements necessary to sit for board certification, and keeps current with the continuing education requirements for that specialty. He keeps current with publications in the field, and has submitted abstracts on his clinical research to the American Thoracic Society.

In its closing brief, the Employer argues that because Dr. Forehand agreed that ventilation perfusion mismatching is generally reversible, the Claimant's disability cannot be due to pneumoconiosis, which is not reversible. The Employer misrepresents Dr. Forehand's testimony: Dr. Forehand stated that ventilation perfusion mismatching could be reversible, and he gave an example of the reversibility of ventilation perfusion defects, where a miner has a low p02 at rest, but their p02 normalizes with exercise. But he clearly testified that this was not the case with the Claimant, whose p02 is normal at rest, but abnormal with exercise. Indeed, according to Dr. Forehand, the Department of Labor specifically instructs physicians to test for this possibility, by performing an exercise study on a miner whose blood gas study is normal at rest, as there are a certain number of miners who will only be impaired with exercise.

When Dr. Hippensteel examined the Claimant, he obtained arterial blood gas study results on exercise that were indicative of "mild hypoxemia." But since the Claimant's diffusion tests were normal, and there were some electrocardiographic changes with exercise, he concluded that this hypoxemia was not pulmonary related, but was cardiac related. Dr. Hippensteel was not willing to concede that the Claimant has pneumoconiosis, but only that there was radiographic evidence for a "possible" diagnosis of pneumoconiosis. He felt that there was no ventilatory or gas exchange impairment that could be ascribed to pneumoconiosis. According to Dr. Hippensteel, the Claimant had evidence of cardiac ischemia with exercise, which was the cause of his hypoxemia.

Dr. Hippensteel pontificated on the need to have a "proper set of data" in order to make "correct conclusions about cause of impairment," characterizing his findings as of "a more complete nature." Dr. Hippensteel concluded that the Claimant had an exercise induced gas exchange impairment related to heart dysfunction, as confirmed by the normal diffusion results. He took Dr. Forehand to task for failing to include cardiac disease as a potential source of the gas exchange impairment, and for using far less data on which to base his conclusions.

However, Dr. Hippensteel's "more complete" findings apparently were not based on a "proper set of data," as the tests performed by Dr. Piriz demonstrated that the Claimant does not have cardiac ischemia with exercise, or any other cardiac impairment. Nevertheless, Dr. Hippensteel continues to cling to his opinion that the Claimant's hypoxemia is not due to lung

disease, but is due to unknown cardiac problems that the tests inexplicably do not show. He speculated that it might be possible to pinpoint these problems with an echocardiogram with exercise, or a blood function test to check for abnormalities with hemoglobin. However, even if these tests produced a normal response, he would feel that there was no answer for the Claimant's abnormal gas exchange.

Although Dr. Hippensteel referred to the concept of a diffusion block, he did not discuss the concept of ventilation perfusion mismatch. Dr. Forehand has acknowledged that the Claimant does not have obstructive airways disease, or a diffusion block (described by Dr. Hippensteel as a relative blockage of a passage of the oxygen across a membrane thickened by interstitial inflammation). Thus, both Dr. Forehand and Dr. Hippensteel agree that the Claimant has a normal diffusion capacity, or DLCO. But as explained by Dr. Forehand, and shown by the 1987 study, hypoxemia caused by exposure to coal dust, or pneumoconiosis, is due to a ventilation perfusion mismatch, not a diffusion block, and would not result in an abnormal DLCO.[6]

In addition to omitting any discussion of ventilation perfusion mismatch as a cause for hypoxemia, Dr. Hippensteel did not refer in any manner to the study cited by Dr. Forehand, although Dr. Forehand indicated that anyone with experience in the field of pulmonary medicine would be familiar with it. Either Dr. Hippensteel is not aware of this study, or he chose not to address it. Neither option reflects favorably on his opinions. Dr. Hippensteel also offered his opinion that less than fifty percent of persons with readings of 2/1 on x-ray have any impairment in their lung function, based on the "medical literature." When pressed, he stated that he was referring to textbooks on occupational medicine. Dr. Hippensteel did not further identify these "textbooks," and Dr. Forehand was not aware of any such literature, studies, or textbooks.

I find that Dr. Hippensteel's shifting conclusions, depending on which set of "complete data" he had before him, are entitled to no weight. I find that his failure to discuss the concept of ventilatory perfusion mismatch, or the 1987 study referenced by Dr. Forehand, are significant and telling omissions. They certainly raise questions of bias and lack of objectivity, questions that I do not need to resolve for purposes of this determination.

Nor do I find Dr. Castle's conclusions, or his lack thereof, to be persuasive evidence on the issue before me. Dr. Castle acknowledged that the Claimant's x-rays were consistent with coal workers' pneumoconiosis. He also concluded, based on the pulmonary function testing, that the Claimant does not have any significant functional abnormality, in the form of airway obstruction, restriction, or diffusion abnormality. He noted the significant drop in p02 with exercise, but he was not able to identify the etiology of the Claimant's exercise induced

[6] In its closing brief, the Employer argues that the study cited by Dr. Forehand is not reliable. Dr. Forehand did not rely on this study in determining that the Claimant's disabling hypoxemia with exercise was due to his pneumoconiosis, or that a ventilation perfusion mismatch causes a drop in the p02 level with exercise; rather, he cited this study to refute Dr. Hippensteel's claim that a normal DLCO necessarily rules out pneumoconiosis as a cause of this drop. I note that the Employer did not take the opportunity to question either of its experts, Dr. Hippensteel and Dr. Castle, about the reliability or applicability of this study. Nor did the Employer question its experts about the medical opinions it offers in its closing brief (for example, the reversibility of the Claimant's perfusion mismatch; the significance of the METS measurement). I find that Dr. Forehand, as an expert in the field of pulmonary medicine, more than adequately established the reliability of this study.

hypoxemia. Dr. Castle was aware of Dr. Hippensteel's initial conclusion that this was due to probable ischemic heart disease, and he felt that the minor changes on EKG during his exercise study of the Claimant "could have" been caused by cardiac changes, although he could not state this with certainty, in the absence of documented cardiac disease. According to Dr. Castle, if pneumoconiosis were the cause of the Claimant's exercise induced hypoxemia, he would expect to find other "abnormalities" on pulmonary testing. Unfortunately, Dr. Castle did not see fit to describe or identify these "other" abnormalities.

I find that Dr. Castle's conclusions, such as they are, are neither well-reasoned nor supported, and I accord them no weight. Dr. Castle apparently did not feel that he had sufficient information to ascribe a cause, including cardiac disease, to the Claimant's exercise induced hypoxemia. There were not the "other abnormalities" on pulmonary function testing that he would expect to see if pneumoconiosis were the cause, but he did not go so far as to actually rule it out. Dr. Castle's report does not really add anything of value: it does not support Dr. Hippensteel's conclusions, nor does it provide a reasoned explanation for the Claimant's exercise induced hypoxemia.[7]

All three physicians agree that the Claimant has radiographic changes consistent with pneumoconiosis; that he has normal pulmonary function, with no evidence of obstruction or restriction; that he has normal diffusing capacity; and that he has normal resting blood gas results, but significant hypoxemia with exercise. Treadmill stress testing, performed by Dr. Piriz, documented no heart disease. Dr. Castle has not offered an opinion on the cause of the Claimant's exercise induced hypoxemia. In the face of the Claimant's normal treadmill test, Dr. Hippensteel continues to attribute his exercise induced hypoxemia to unknown cardiac disease. As discussed above, I have accorded no weight to Dr. Hippensteel's opinions.

I place determinative weight on the opinions of Dr. Forehand. I found Dr. Forehand's discussion of the different types and workings of hypoxemia to be thorough and detailed. His opinions are based on his extensive experience and training in the field of pulmonary medicine, his thorough examination and testing of the Claimant, and his review and consideration of the totality of the medical evidence of record.[8] His conclusions are in accord with the objective test results, and are supported by a medical study in a respected publication.[9] I find Dr. Forehand's opinions to be thoroughly reasoned and supported by the medical evidence of record, and I accord them determinative weight. Accordingly, I find that the Claimant has established that he is totally disabled, pursuant to § 718.204, and that his total disability is due to pneumoconiosis.

## CONCLUSION

[7] Dr. Castle's report is very cautiously and carefully worded: he did not agree with Dr. Hippensteel that the Claimant's exercise induced hypoxemia was due to probable ischemic heart disease. Rather, he stated that the minor ST-T wave changes on EKG during the exercise study could possibly have been caused by cardiac changes.

[8] I do not accept the Employer's characterization of Dr. Forehand's testimony as "merely suggesting that a study might have shown or support a connection between his hypoxemia and pneumoconiosis."

[9] In contrast, Dr. Hippensteel has offered no scientific support for his conclusions and, according to Dr. Forehand, the "medical literature" to which he referred does not exist.

The Claimant has established by a preponderance of the medical evidence that he has pneumoconiosis, and that he is totally disabled due to pneumoconiosis, He is therefore entitled to benefits under the Act.

**ORDER**

It is ordered that the claim of James C. Honaker for benefits under the Black Lung Benefits Act is hereby GRANTED.

It is further ordered that the Employer, Sea B Mining Company, shall pay to the Claimant all benefits to which he is entitled under the Act beginning in November, 2002.

SO ORDERED.

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LINDA S. CHAPMAN
Administrative Law Judge

**ATTORNEY'S FEES**

An application by Claimant's attorney for approval of a fee has not been received. Thirty days is hereby allowed to Claimant's counsel for submission of such an application. A service sheet showing that service has been made upon all the parties, including the claimant, must accompany the application. The parties have ten days following receipt of any such application within which to file any objections. The Act prohibits the charging of a fee in the absence of an approved application.

**NOTICE OF APPEAL RIGHTS**: Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 (thirty) days from the date of this decision, by filing a Notice of Appeal with the Benefits Review Board, P.O. Box 37601, Washington, D.C. 20013-7601. *A copy of a Notice of Appeal must also be served on Donald S. Shire, Esq., Associate Solicitor for Black Lung Benefits, 200 Constitution Avenue, NW, Room N-2117, Washington, D.C. 2021*